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FILE 'MEDLINE' ENTERED AT 07:35:56 ON 05 NOV 2003

L1	12 S REVIEW AND HEME(15W) BIND?
L2	339 S REDOX(25W) HEME
L3	4 S L2 AND REVIEW
L4	888 S REDOX(10W) PROTEIN
L5	23 S L4 AND REVIEW
L6	3 S L5 AND HEME
L7	35 S L4 AND HEME(15W) PROTEIN

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L7 ANSWER 16 OF 35 MEDLINE on STN
 AN 1999371536 MEDLINE
 DN 99371536 PubMed ID: 10443936
 TI Expression, purification, and biochemical characterization of SAG, a ring
 finger **redox**-sensitive **protein**.
 AU Swaroop M; Bian J; Aviram M; Duan H; Bisgaier C L; Loo J A; Sun Y
 CS Department of Molecular Biology, Parke-Davis Pharmaceutical Research,
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 SO FREE RADICAL BIOLOGY AND MEDICINE, (1999 Jul) 27 (1-2) 193-202.
 Journal code: 8709159. ISSN: 0891-5849.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199911
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 Last Updated on STN: 20030102
 Entered Medline: 19991112
 AB We recently reported the cloning and characterization of SAG (sensitive to
 apoptosis gene), a novel zinc RING finger protein, that is redox
 responsive and protects mammalian cells from apoptosis. Here we report
 the expression, purification, and biochemical characterization of SAG.
 Bacterially expressed SAG is brown in color and dithiothreitol
 (DTT)-sensitive. SAG forms large oligomers without DTT that can be
 reduced into a monomer in the presence of DTT. These features help us to
 purify SAG using the chromatography with or without DTT. Likewise,
 purified SAG is redox sensitive. Upon H2O2 exposure, SAG forms oligomers
 as well as monomer doublets due to the formation of the inter- or
 intramolecular disulfide bonds, respectively. This process can be
 reversed by DTT or prevented by pretreatment with the alkylating reagent,
 N-ethylmaleimide (NEM). Although SAG contains two putative **heme**
 -binding sites and a RING finger domain, the **protein** appears not
 to bind with heme and to lack transcription factor activity as determined
 in a Gal4-fusion/transactivation assay. Wildtype, but not RING finger
 domain-disrupted SAG mutants, prevents copper-induced lipid peroxidation.
 These results, along with our previous observations, suggest that SAG is
 an intracellular antioxidant molecule that may act as a redox sensor to
 buffer oxidative-stress induced damage.

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